STN Search History

FILE 'HOME' ENTERED AT 10:27:26 ON 27 JUL 2002

=> index bioscience, medicine

=> s (PDD or (pervasive (a) develop#####) or parkinson or nuerologic or dysautonomic or dysautonomia) and (fec### or stool)

L1 QUE (PDD OR (PERVASIVE (A) DEVELOP#####) OR PARKINSON OR NUEROLOGIC OR DYS AUTONOMIC OR DYSAUTONOMIA) AND (FEC### OR STOOL)

=> d rank

F1	370	USPATFULL
F2	52	MEDLINE
F3	45	SCISEARCH
F4	39	EMBASE
F5	29	PROMT
F6	27	BIOSIS
F7	26	CAPLUS
F8	26	DRUGU
F9	24	TOXCENTER
F10	16	NLDB
F11	15	PASCAL
F12	11	DDFU
F13	11	FEDRIP
F14	9	WPIDS
F15	9	WPINDEX
F16	8	JICST-EPLUS
F17	8	USPAT2
F18	5	ESBIOBASE
F19	4	ADISNEWS
F20	4	IFIPAT
F21	3	CANCERLIT
F22	3	LIFESCI
F23	2	ADISALERTS
F24	2	BIOTECHNO
F25	2	DDFB
F26	2	DRUGB
F27	1	AGRICOLA
F28	1	CABA
F29	1	CIN
F30	1	CROPU
F31	1	NIOSHTIC
F32	1	PHIN

=> file medline, scisearch, embase, promt, biosis, caplus, drugu, toxcenter, pascal

L2	257	L1
L3	150	DUP REMOVE L2 (107 DUPLICATES REMOVED)
L4	3	L3 AND (HELICOBACTER OR PYLORI)
L5	8	L3 AND (PATHOGEN OR ANTIGEN)
L6	17	L3 AND (PDD OR (PERVASIVE (A) DEVELOP#####) OR DYSAUTONOMIC OR DYSAUTONOMIA)
L7	16	L6 NOT L4
L8	5	L3 AND (PDD OR (PERVASIVE (A) DEVELOP#####))

L7 ANSWER 1 OF 16 MEDLINE
AN 2000062067 MEDLINE
DN 20062067 PubMed ID: 10596931
TI The association of Clostridium botulinum type C with equine grass sickness: a toxicoinfection?.
CM Comment in: Equine Vet J. 1999 Nov;31(6):451-2
AU Hunter L C; Miller J K; Poxton I R
CS Department of Medical Microbiology, University of Edinburgh Medical School, Scotland.
SO EQUINE VETERINARY JOURNAL, (1999 Nov) 31 (6) 492-9.
Journal code: 0173320. ISSN: 0425-1644.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200002
ED Entered STN: 20000309
Last Updated on STN: 20000309
Entered Medline: 20000224
AB The cause of grass sickness, an equine **dysautonomia**, is unknown. The disease usually results in death. Gastrointestinal (GI) dysfunction is a common clinical manifestation in all forms of the disease. It is generally thought that equine grass sickness (EGS) is caused by an ingested or enterically produced neurotoxin which is absorbed through the GI tract. Clostridium botulinum was first implicated as a causative agent when it was isolated from the GI tract of a horse with EGS in 1919. The aim of the present study was to investigate the hypothesis that EGS results from toxicoinfection with C. botulinum type C: growth of the bacterium in the GI tract with production of toxin (BoNT/C). Ileum contents and faeces from horses with EGS were investigated for BoNT/C, and indirectly for the presence of C. botulinum type C, and compared with control samples from horses without EGS. BoNT/C was detected directly by ELISA in the ileum of 45% (13/29) of horses with EGS compared to 4% (1/28) of controls, and in the faeces of 44% (20/45) of horses with EGS compared to 4% (3/77) of controls. Levels of up to 10 Mlg toxin/g wet weight of gut contents were observed. The one control horse with detectable toxin in the ileum had been clinically diagnosed as having acute EGS, but this was not confirmed by histopathology. The organism was detected indirectly by assaying for BoNT/C by ELISA after enrichment in culture medium. C. botulinum type C was shown to be present in 48% (14/29) of ileum samples and 44% (20/45) of faecal samples from horses with EGS, compared with 7% (2/27) of ileum samples and 8% (6/72) of faecal samples from controls. These results support the hypothesis that EGS results from a C. botulinum type C toxicoinfection.

L7 ANSWER 2 OF 16 MEDLINE
AN 2000039166 MEDLINE
DN 20039166 PubMed ID: 10572871
TI Cecal impaction due to **dysautonomia** in a llama (Lama glama).
AU Kik M J; van der Hage M H
CS Department of Veterinary Pathology, Utrecht University, The Netherlands.
SO JOURNAL OF ZOO AND WILDLIFE MEDICINE, (1999 Sep) 30 (3) 435-8.
Journal code: 8915208. ISSN: 1042-7260.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200001
ED Entered STN: 20000114

Last Updated on STN: 20000114
Entered Medline: 20000106

AB A llama (Lama glama) died after 1 wk of obstipation, lethargy, and rolling. Necropsy showed that the stomach and small intestine were distended with gas and fluid. The cecum was impacted with dry contents and the colon was empty. No gross lesions were found in the wall of the gastrointestinal tract or other organs. Histologic changes consisted of chromatolysis of neurons of autonomic ganglia, enteric plexi, and the accessory cuneate nucleus, consistent with lesions associated with **dysautonomia** in other domestic animals.

L7 ANSWER 3 OF 16 MEDLINE
AN 94164498 MEDLINE
DN 94164498 PubMed ID: 8119549
TI Diarrhea and autonomic dysfunction in a patient with hexosaminidase B deficiency (Sandhoff disease).
AU Modigliani R; Lemann M; Melancon S B; Mikol J; Potier M; Salmeron M; Said G; Poitras P
CS Department of Gastroenterology, Hopital St-Louis, Paris, France.
SO GASTROENTEROLOGY, (1994 Mar) 106 (3) 775-81.
Journal code: 0374630. ISSN: 0016-5085.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199404
ED Entered STN: 19940412
Last Updated on STN: 19940412
Entered Medline: 19940401

AB The causal factors and the physiopathology of motor diarrhea are still unclear. This case report describes a 60-year-old white man with severe diarrhea for more than 10 years and minor signs of autonomic dysfunction. Extensive investigation showed that small intestinal motility and absorption were normal but that accelerated colon transit precluded water and solute absorption from the large bowel. Orthostatic hypotension, sexual dysfunction, and loss of sweating suggested dysfunction of the autonomous nervous system, which was confirmed by reduced plasma concentrations of norepinephrine and dopamine. Rectal biopsy specimens showed enlarged enteric ganglion cells filled with lipidic material. Levels of total hexosaminidase and hexosaminidase B in plasma, white blood cells, and fibroblasts were decreased, as found in Sandhoff disease. The pedigree of the proband's family showed several affected and heterozygous individuals, detected by examination of total hexosaminidase and hexosaminidase B levels in plasma. Among the five homozygous subjects, three had a clinical picture of diarrhea and orthostatic hypotension since the age of 50. Therefore, hexosaminidase B deficiency should probably be regarded as a cause for **dysautonomia**; dysfunction of the gastrointestinal tract, manifested by motor diarrhea or esophageal dysmotility, could be the initial and prevalent presentation of **dysautonomia**.

L7 ANSWER 4 OF 16 MEDLINE
AN 74279929 MEDLINE
DN 74279929 PubMed ID: 4844135
TI Basis of nocturnal polyuria in patients with autonomic failure.
AU Wilcox C S; Aminoff M J; Penn W
SO JOURNAL OF NEUROLOGY, NEUROSURGERY AND PSYCHIATRY, (1974 Jun) 37 (6) 677-84.
Journal code: 2985191R. ISSN: 0022-3050.
Report No.: NASA-74279929.

CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Space Life Sciences
EM 197410
ED Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19741009

L7 ANSWER 7 OF 16 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 93:412994 SCISEARCH
GA The Genuine Article (R) Number: LJ792
TI SYNCOPE IN NEUROLOGICAL DISEASES
AU DAFFERTSHOFER M; HENNERICI M (Reprint)
CS UNIV HEIDELBERG, KLINIKUM MANNHEIM, NEUROL KLIN, POSTFACH 100023, D-68135 MANNHEIM 1, GERMANY
CYA GERMANY
SO HERZ, (JUN 1993) Vol. 18, No. 3, pp. 187-201.
ISSN: 0340-9937.
DT Article; Journal
FS CLIN
LA German
REC Reference Count: 82
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Transient loss of consciousness due to an acute decrease in cerebral blood flow is the classical but not commonly accepted definition of syncope. Besides cardiac or respiratory induced syncopes, various neurological causes affecting the autonomic pathways, which are involved in maintaining cerebral autoregulation, could lead to syncope. The most common form is the simple fainting attack seen in young people (15 to 20%). Special forms of vasovagal syncopes are the micturition and swallowing syncopes.

Usually there is some warning, including weakness, sweating, pallor, nausea, yawning, sighing, hyperventilation, blurred vision, impaired external awareness, and dilation of pupils, followed by unconsciousness with pallor, coldness of the skin and sweating. At the onset of unconsciousness, the pulse is usually imperceptible; when it returns it is slow. Like most non-cardiac syncopes, vasovagal syncopes are often associated with a specific trigger mechanism such as pain, fear, emotional reactions, injury, surgical manipulation, and an upright position. Orthostasis is the main trigger for syncope, and nearly every syncope appears while the patient is standing or at least sitting.

While the autonomic nervous system in vasovagal syncopes is physiologically intact, areflexic syncope results from either functional or structural lesions of the autonomic nervous system. Pathophysiologically, an insufficient compensatory increase in heart rate, cardiac output, and arteriolar vasoconstriction are due to a disfunction of the orthostatic cerebrovascular autoregulation. Impairment of autonomic function due to a variety of lesions involving the autonomic reticular system, including syringobulbia, posterior fossa tumors, ischemia, and inflammatory diseases, leads to blood pressure dysregulation. In general, spinal cord transsection produces postural hypotension if the lesion is above the T6 level. Intramedullary and extramedullary tumors, transverse myelitis and syringomyelia involving the cord above T6 level may also produce autonomic failure and syncope. In patients with polyneuropathy, autonomic involvement is not uncommon. It is particularly conspicuous in diabetic neuropathy, and insulin treatment may further contribute to the severity of postural hypotension. Autonomic involvement in Guillain-Barre syndrome leads to orthostatic hypotension and may be fatal. sometimes due to cardiac arrhythmia or asystole. Other neuropathies leading to

orthostatic hypotension and syncope include metabolic, autoimmune, hereditary, toxic. and inflammatory neuropathies. Impairment in Wernicke's encephalopathy may be related to central or peripheral involvement. The extent, to which autonomic function, and particularly cardiovascular regulation is impaired in **Parkinson**'s disease, is disputed. but clinical data evidenced a higher probability for syncope. In other neurological diseases like the Shy-Drager syndrome, patients with multiple system atrophy, pandysautonomia, and idiopathic orthostatic hypotension, syncopes are the leading symptom.

The primary differential diagnosis of syncope must be made to epilepsy. In many cases the distinction between syncope and epilepsy is an easy one when a detailed history is available. Limpness, pallor, and sweating during unconsciousness are much more characteristic of syncope than epilepsy. The duration of a syncopal attack is relatively short, and a patient is usually mentally clear on regaining consciousness. Incontinence of urine sometimes occurs in syncopal attacks, but **fecal** incontinence is exceedingly rare, if it occurs at all. Difficulty in diagnosis may arise if the onset of the attack is sudden and if there are convulsive movements during the period of unconsciousness. In the absence of a detailed report of clinical signs, the instrumental work-up may often be rather extensive including EEG monitoring studies during wakefullness and sleep. In the case of specific epileptic alterations an epileptic attack is very probable while a normal or unspecific abnormal EEG cannot be used for differential diagnosis. A single orthostatic testing (Schellong's test) can uncover orthostatic hypotension suggesting syncope. However, the recently introduced combined registration of heart rate and blood pressure with measurement of the cerebral blood flow by transcranial Doppler is particularly prognostic for the detection of cerebrovascular dysregulation in the presence of normal systemic blood pressure and heart rate. Nevertheless. some attacks of unconsciousness with convulsive movements remain unclear: Some of them have recently been classified as convulsive syncopes. Physiologically, it can be assumed that either cerebral hypoxia (e.g. during a syncope) could induce epileptic alterations or the other way around, that epilepsy with consecutive cerebral hypoxia could lead to this syncope syndrome. In these cases, a clear differentiation between syncope and epilepsy may not be possible, but treatment in both directions may be worth a trial.

L7 ANSWER 9 OF 16 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 2000107657 EMBASE
TI Autonomic dysfunction in MS.
AU Frontoni M.; Giubilei F.
CS Dr. M. Frontoni, I Clinica Neurologica, Viale dell'Universita' 30, 00185 Rome, Italy
SO International MS Journal, (2000) 6/3 (78-87).
Refs: 73
ISSN: 1352-8963 CODEN: IMSJFO
CY United Kingdom
DT Journal; General Review
FS 005 General Pathology and Pathological Anatomy
006 Internal Medicine
008 Neurology and Neurosurgery
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English; French; German
AB As a central nervous system disease characterized by disseminated, multifocal lesions, multiple sclerosis (MS) can generate a variety of symptoms, including those related to the involvement of autonomic functions. **Dysautonomia** is often a serious problem in the

disease owing to its disabling effects. Autonomic disturbances, such as bladder, bowel and sexual dysfunction, as well as cardiovascular and sweating abnormalities, occur with varying frequency in the course of MS. This article reviews the prevalence, clinical expression and management of autonomic disturbances, and discusses the underlying anatomophysiological mechanisms, as well as the possible correlations between symptoms and lesion localizations. The investigations for detection and evaluation of specific autonomic dysfunction are also considered.

ANSWER 10 OF 16 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 97101943 EMBASE
DN 1997101943
TI Gastrointestinal dysfunction in autonomic neuropathy.
AU Chelimsky G.; Wszolek Z.; Chelimsky T.C.
CS Dr. G. Chelimsky, Department of Neurology, Case Western Reserve University, School of Medicine, Cleveland, OH, United States
SO Seminars in Neurology, (1996) 16/3 (259-268).
Refs: 85
ISSN: 0271-8235 CODEN: SEMNEP
CY United States
DT Journal; General Review
FS 008 Neurology and Neurosurgery
022 Human Genetics
048 Gastroenterology
LA English

L7 ANSWER 11 OF 16 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 95226724 EMBASE
DN 1995226724
TI HTLV-1 associated pandysautonomia with adrenal dysfunction [2].
AU Ando Y.; Ando E.; Vchino M.; Ando M.
CS First Dept. of Internal Medicine, Kumamoto University Sch. of Medicine, 1-1-1 Honjo,Kumamoto 860, Japan
SO Muscle and Nerve, (1995) 18/8 (928-929).
ISSN: 0148-639X CODEN: MUNEDE
CY United States
DT Journal; Letter
FS 007 Pediatrics and Pediatric Surgery
008 Neurology and Neurosurgery
LA English

L7 ANSWER 13 OF 16 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1986:37053 BIOSIS
DN BR30:37053
TI FELINE **DYSAUTONOMIA**.
AU GASKELL C J; SHARP N J H
CS DEP. OF VETERINARY CLINICAL SCIENCES, UNIV. OF LIVERPOOL.
SO 2ND MEETING OF THE CLINICAL AUTONOMIC RESEARCH SOCIETY, LONDON, ENGLAND, NOV. 16, 1984. J AUTON NERV SYST. (1985) 14 (1), 100.
CODEN: JASYDS. ISSN: 0165-1838.
DT Conference
FS BR; OLD
LA English

L7 ANSWER 16 OF 16 PASCAL COPYRIGHT 2002 INIST-CNRS. ALL RIGHTS RESERVED.
AN 2000-0008040 PASCAL

TIEN Season of birth in autism : A fiction revisited
AU LANDAU E. C.; CICCHETTI D. V.; KLIN A.; VOLKMAR F. R.
CS Bar Ilan University, Ramat Gan, Israel; Child Study Center-Yale University, PO Box 207900, New Haven, Connecticut 06520, United States
SO Journal of autism and developmental disorders, (1999), 29(5), 385-393, 37 refs.
ISSN: 0162-3257 CODEN: JADDDQ
DT Journal
BL Analytic
CY United States
LA English
AV INIST-15018, 354000080765730050

AB Variations of season of birth among autistic individuals were studied. The replicability of previously reported increases in birth rates in the months of March and August were examined in groups of individuals with autism or mental retardation (the comparison group). The sample was obtained from the Yale Child Study Center Developmental Disabilities Clinic and from the DSM-IV Autism/**PDD** field trial. Data were analyzed by applying the Jonckheere test of ordinal trend and the chi-square test, with Yates correction factor. With respect to March and August births, and with calculations based on the beginning and middle of the month, no significant seasonal effect was observed. Samples were subcategorized into verbal and mute groups, and again results failed to support the seasonality hypothesis.

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Gut

☐ **Gut** 43: 285-287. [Abstract] [Full Text] [PDF]
Ischaemic enterocolitis complicating idiopathic **dysautonomia**
J M Woodward, D S A Sanders, M R Keighley, and R N Allan

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To see an article, click its [Full Text] link. **To review many abstracts**, check the boxes to the left of the titles you want, and click the 'Get All Checked Abstract(s)' button. **To see one abstract at a time**, click its [Abstract] link.

Search Criteria:

Title/Abstract: dysautonomia or dysautonomic or "pervasive development"
Anywhere in Article: (bacteria or pathogen) and (fecal or stool)
In Journals: J. Exp. Med., J. Exp. Med., Am. J. Respir. Cell Mol. Bio., Annu. Rev. Biochem., Annu. Rev. Biomed. Eng., Annu. Rev. Biophys. Biomol. Struct., Annu. Rev. Cell. Dev. Biol., Annu. Rev. Genet., Am J Physiol Cell Physiol, Am J Physiol Lung Cell Mol Physiol, Bioinformatics, Biol. Reprod., Biophys. J., EMBO J., EMBO Rep., Eur. J. Biochem., FASEB J, Genetics, Genes & Dev., Genome Res., Glycobiology, Hum. Mol. Genet., J. Biol. Chem, J. Cell Biol., J. Clin. Invest., J. Histochem. Cytochem., J. Lipid Res., Mol. Biol. Cell, Mol. Biol. Evol., Mol. Cell. Biol., Mol. Pathol., Mol. Pharmacol., Mutagenesis, Nucleic Acids Res., Physiol Genomics, PLANT CELL, PNAS, Protein Eng., Science, Antimicrob. Agents Chemother., Appl. Envir. Microbiol., Annu. Rev. Micrbiol., Clin. Microbiol. Rev., Genes & Dev., Infect. Immun., Int J Syst Evol Microbiol, J. Antimicrob. Chemother., J. Bacteriol., J. Clin. Microbiol., J. Gen. Virol., J. Virol., Microbiology, Microbiol. Mol. Biol. Rev., PNAS, Science, Annu. Rev. Immunol., Clin. Diagn. Lab. Immunol., Infect. Immun., Int. Immunol., J. Clin. Invest., J. Exp. Med., PNAS, Science, Annu. Rev. Neurosci., Brain, Cereb Cortex, Chem Senses, Genes & Dev., J. Cogn. Neurosci., J. Neurochem., J Neurophysiol, J. Neuropsychiatry. Clin. Neurosci., J. Neurosci., Learn.

Mem., Neural Comput., PNAS, Science, AAP News, Acad. Emerg. Med., Acad. Med., Age Ageing, Alcohol Alcohol., Am. J. Clinical Nutrition, Am. J. Respir. Crit. Care Med., Am. J. Roentgenol., Anesth. Analg., Ann. Rheum. Dis, Annu. Rev. Med., Annu. Rev. Nutr., Annu. Rev. Public Health., Arch. Dis. Child., BMJ, Br. J. Anaesth., J. Orthod., Br. J. Ophthalmol., Br. J. Sports Med., Fam. Pract., Health Educ. Res., Health Policy Plan., Health Promot. Int., Int. J. Epidemiol., Invest. Ophthalmol. Vis. Sci., J. Clin. Pathol., J. Deaf Stud. Deaf Educ., J. Epidemiol. Community Health, J. Med. Ethics, Med. Humanit., NeoReviews, Nephrol. Dial. Transplant., N. Engl. J. Med., J. Nutr., Occup. Environ. Med., Ophthalmology, Pediatr. Res., Pediatrics, Pediatr. Rev., Postgrad. Med. J., QJM, Qual. Saf. Health Care, Rheumatology, Sex. Transm. Inf., Tob. Control, Oncologist, Am. J. Pathol., Am J Physiol Gastrointest Liver Physiol, Am. J. Roentgenol., Anesth. Analg., Annu. Rev. Pharmacol. Toxicol., Biol. Reprod., Blood, Br. J. Pharmacol., BMJ, Carcinogenesis, Clin. Chem., Clin. Diagn. Lab. Immunol., Drug Metab. Dispos., Gut, Hum. Reprod., J. Am. Soc. Nephrol., J. Am. Med. Inform. Assoc., J. Clin. Oncol., J. Exp. Med., J. Clin. Invest., J. Invest. Dermatol., J. Med. Genet., J Natl Cancer Inst, J Natl Cancer Inst Monographs, J. Pharmacol. Exp. Ther., Mol. Hum. Reprod., N. Engl. J. Med., Obes. Res., Pharmacol. Rev., PNAS, Experimental Biology and Medicine, RadioGraphics, Radiology, Science, Stem Cells, Transfusion, Toxicol. Sci.

WEST Search History

DATE: Saturday, July 27, 2002

Set Name side by side	Query	Hit Count	Set Name result set
	DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=OR		
L10	L9 not fallon.in.	0	L10
L9	((pervasive adj development) dysautonomic dysautonomia) same (stool fec$4)	4	L9
L8	L6 and (fec$3 stool)	3	L8
L7	L6 and (fec$3 stool) adj sample	3	L7
L6	((pervasive adj development) dysautonomic dysautonomia) same (assay detect marker immunoassay analy$4)	30	L6
L5	((pervasive adj development) dysautonomic dysautonomia) same (pathogen bacteria) same (assay detect marker immunoassay analy$4)	1	L5
L4	((pervasive adj development) dysautonomic dysautonomia) same ((pathogen bacteria) ((stool fecal) with sample same (assay detect marker immunoassay analy$4)))	4	L4
L3	L1 and @ad<20001116	9	L3
L2	L1 and 20001116	1	L2
L1	(pdd (pervasive adj development) dysautonomic dysautonomia) same ((pathogen bacteria) ((stool fecal) with sample same (assay detect marker immunoassay analy$4)))	12	L1

END OF SEARCH HISTORY